Chronic Low Back Pain - A Summary and Review

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Abstract: A review of the etiology, clinical, radiological and laboratory presentation, differential diagnosis and management goals of chronic low back pain is presented.

Index Terms: Low Back Pain, Chronic, Chiropractic.

Summary: Chronic low back pain (CLBP) is defined as low back pain lasting greater than six months or regular intermittent low back pain attacks over more than a one year period. The presence of a treatable lesion has usually been eliminated and pain has become the patients main complaint (2).

Only 5% of low back pain attacks progress to the chronic stage, but this subset of patients is responsible for 85% of the costs associated with low back pain (1).

Low back pain develops because of irritation of structures sensitive to pain ie. disc, joints, nerves, muscles, ligaments, blood vessels and bone (2,4).

It is difficult to identify precisely the origin of CLBP, because even if its characteristics point to a given structure, the pain often remains unspecific. Additionally, histologically there is often no tissue modification (2).

The influence of psychologic and social factors on the continuation of CLBP is well recognised (3).

It is important to look briefly at the potential sources of pain:

1. Disc: The disc can be a source of pain by both mechanical or chemical means. Further, it can either manifest a lesion in a contained or non-contained manner. Cracks or fissures in the annulus may produce pain (contained disc lesion) or alternatively disc material can rupture into the vertebral body (schmorl's node) or into the neural canal (disc prolapse) producing chemical and mechanical irritation, this is known as a non-contained disc lesion (5).

2. Facet Joints: The facet joints may be a source of pain from inherit degeneration, inflammation or dysfunction (6), or by osteoarthritic and hypertrophic changes causing stenosis (7).

3. Nerve: The spinal nerve root is sensitive to mechanical pressure and chemical irritation eg. from a disc protrusion, surrounding stenosis or leaking nuclear material (5) (7) (8).

The root itself is different from a peripheral nerve in that it has no epineurium to resist mechanical stresses. Rather, it is encased in a flimsy gauze-like pia through which cerebrospinal fluid moves to supply the majority of the nerves nutrition (9).

According to Mooney, inflammation and fibrosis could readily obliterate this source of nutrition. He also states that mechanical events such as vibration cause the release of Substance P (a neurotransmitter in the dorsal root ganglion) converting this mechanical event into a chemical one and thereby facilitating pain (9).

4. Muscles: Frymoyer and Gordon (1) claim that the contribution of paraspinal muscles to low back disease requires more study, despite the fact that "low back strain" remains a common diagnosis.

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The presence of "trigger points" in the muscles of CLBP patients is documented (10) and the clinical syndrome of fibrositis syndrome with its accompanying sleep disturbance and proximal muscle tightness is gaining credence (11).

5. Ligaments: Spinal ligaments play an important role in at least four types of spinal pain: spinal stenosis, injury, degenerative lesions and segmental instability (12).

6. Blood Vessels: The mechanical squeezing of blood vessels supplying the nerves causes ischaemia in certain areas and subsequent distension in others creating a chemical imbalance to nerve roots (9).

This can cause spontaneous neural activity and make the nerves more sensitive to mechanical stimulation (9).

7. Bone: Apart from direct pathology or trauma such as fracture the notion that vertebral bodies can cause pain has not been tested (14). It is presumed that the pain of spinal osteoporosis arises from the vertebral bodies but this also has not been proved (4).

Interestingly, a significant fraction (21%) of patients with bone metastases do not report bone pain.

Differential Diagnosis:

Chronic low back pain describes a symptom not a diagnosis, therefore all known causes of low back pain make up the differential list. Apart from disorders of the lumbar spine itself attention should be given to visceral or vascular disease in the abdomen or pelvis.

However, any structure in the region with a nerve supply is potentially a source of pain. Wiesel et al found that 14 patients in a trial involving 109 chronic low back pain patients had a major underlying medical problem (14).

Usual and Customary Examination procedures:

Physical Examination:

Spinal mobility measurements, like physical measurements in general have only moderate relationships with the degree of CLBP (15). Rotation and lateral flexion correlate better with CLBP than do forward flexion and extension (15).

A number of methods have been developed to assess the CLBP patient. These methods usually combine a number of physiological and psychological criteria. For instance Million et al developed a method for measuring progress in back pain patients using a "global index" of symptoms and an objective measure of spinal motion (16).

While Kagan and Evans (17) devised a functional rating scale (FRS) to quantify the patients level of activity and relative personal independence. The FRS addresses six separate areas of independent behaviour: vocational activity, activities of daily living, time spent in bed, medication usage, dependence on aids and usage of TENS.

Another useful index of disability in CLBP is the OWESTRY low back pain disability questionnaire (18) which looks at 10 parameters:

1. Pain intensity
2. Personal care (washing, dressing)
3. Lifting
4. Walking
5. Sitting
6. Standing
7. Sleeping
8. Sex life
9. Social life
10. Travelling

Pain drawings are also useful in detecting non-organic factors in CLBP (19).

Regardless of the measurement systems used they do not replace a thorough history and physical examination.

Diagnostic Imaging:

Radiology has become a valuable and integral part of the evaluation of the CLBP patient particularly in the areas of pathology and biomechanics.

The diagnostic imaging of choice in the CLBP patient will be determined by many factors, however there are few patients who will not have had plain films and a CT scan. these two imaging modalities give a baseline of positive and negative diagnostic information for the differential diagnosis of CLBP.

Other tests that may be employed are:

Discogram, Myelogram, Bone Scan, Tomography, Epidural Venography, MRI and Cineradiography and combinations thereof.

The most common conditions which may be linked to CLBP and found by these imaging modalities are:

1. Spondyloarthropathy
2. Internal disc disruption
3. Disc herniation or protrusion
4. Sequestrated disc fragment
5. Lateral and central stenosis
6. Infection
7. Fibrosis/Scar
8. Arachnoiditis
9. Pseudarthrosis
10. Discogenic spondylosis
11. Facet arthropathy
12. Segmental instability
13. Neoplasm

Clinical Laboratory Tests:

Most patients with CLBP should have the following baseline blood tests:

1. Full blood count
2. Erythrocyte Sedimentation Rate or C-reactive protein
3. Rheumatoid factor
4. HLA-B27
5. Serum Urate
6. Anti nuclear factor
7. Alkaline Phosphatase
8. Acid phosphatase (in males)

This will usually eliminate a number of potential sources of CLBP including rheumatoid arthritis, ankylosing spondylitis, gout, bony metastases and infection.

EMG and Nerve Conduction Studies: EMG will confirm the presence of impaired nerve function in chronic pain (20). Saal states that electrophysiologic tests are useful in stenosis, intraneural fibrosis (scar), occasionally instability and reoccurrent disc protrusion, all of which are potential sources of CLBP (21).

Goals of Treatment:

1. Reduce Pain and Inflammation
2. Restore Function
3. Prevent Reocurrence

Chiropractic Management:

1. In terms of pain and inflammation reduction the chiropractor use a number of methods including physiotherapeutic modalities, acupuncture, trigger point therapy, mobilisation, manipulation and even analgesics and anti-inflammatory drugs.

2. However, a major role for the chiropractor in the management of CLBP can develop in the area of functional restoration. Hazard et al (22) tested a treatment program modelled after Mayer et al (23) where patients have an initial intensive 3 weeks of physical, psychological and educational sessions including work hardening.

Thereafter, they are seen on a follow up treatment program 1.5 to 2 days per week for an average of 3 weeks.

The results on 90 patients studied showed 59 patients completed the program, 5 dropped out, 17 were denied access by their insurers and there were 6 crossover patients, 3 others were admitted but refused treatment.

At the end of 1 year 81% of the program graduates, 40% of the dropouts and 29% of those denied access to the program had returned to work.

Clearly, there is a place for functional restoration in the management of the CLBP patient, and it is important that the chiropractor assumes a leading role in the multi-disciplinary team approach to CLBP.

Prognosis:

According to White (24) the prognosis for CLBP patients is dismal. Fewer than 50% of those disabled greater than 6 months ever return to work and for those disabled for greater than 2 years re-employment is virtually nil (24). With the advent in recent times of functional restoration programmes the outlook may not be so dismal.

Chronic Low Back Pain as a Complication:

Below is a list of conditions which are possible or potential sources of CLBP, it is substantial but not exhaustive: (1,3,5,6,7,8,9,10,11,24,25,26,27)

1. Disc degeneration
2. Disc herniation
3. Spinal stenosis
4. Segmental instability
5. Facet degeneration
6. Spondyloarthropathy
7. Neoplasm
8. Infection
9. Fibrosis or scar formation (post-surgical)
10. Arachnoiditis (post-surgical)
11. Reflex Sympathetic Dystrophy (post-surgical)
12. Meningocele (post-surgical)
13. Psychological causes
14. Alcoholism/Smoking
15. Pagets disease/Osteomalacia/Osteporosis
16. De-conditioning
17. Myofascial pain syndromes
18. Baastrup's syndrome
19. Stress fracture/spondylolysis/spondylolisthesis
20. Scheuermann's disease
21. Discogenic spondylosis
22. Anomalies, scoliosis, pelvic obliquity
23. Trauma such as compression fracture
24. Sacro-iliac joint syndrome
25. Idiopathic

REFERENCES

1. Frymoyer J.W., Gordon S.L. Research Perspectives in Low Back Pain. Spine. 14:12, 1384-1390, 1989.

2. LeBlanc F.E. (ed) Scientific Approach to the Assessment and Management of Activity-Related Spinal Disorders. Report of the Quebec Task Force on Spinal Disorders. Spine. 12:7S, S16-S21, 1987.

3. Waddell G. et al Chronic Low-Back Pain, Psychologic Distress, and Illness Behaviour. Spine 9:209-213, 1984.

4. Bogduk N., Twomey L.T. Clinical Anatomy of the Lumbar Spine. Churchill Livingstone. 1987. pp. 130-138.

5. (4) p 139-147.

6. Mooney V., Roberston J. The Facet Syndrome. Clin. Orthop. 115:149-156, 1976.

7. Ciric I et al. The Lateral Recess Syndrome., J. Neurosurg. 53:433-443, 1980.

8. Mixter W.J., Barr J.S. Rupture of the Intervertebral Disc with involvement of the Spinal Canal. N.Engl.J.Med. 211:210, 1934.

9. Mooney V. Where is the Pain Coming From? Spine.12:8, 1987. 754-759.

10. Melzack R. et al Trigger Points and Acupuncture Points for Pain: Correlations and Implications. Pain 1977, 3(1):3-23.

11. Reilly P.A., Littlejohn G.O. Fibrositis/Fibromyalgia Syndrome: The Key to the Puzzle of Chronic Pain. Med.J.Aust.March 5, 1990. Vol 152. pp. 226-228.

12. Frymoyer J.W., Gordon S.L. New Perspectives on Low Back Pain. Amer. Acad of Orthop. Surg. Symposium. May 1988. Illinois. p. 232.

13. Palme E. et al. Pain as an Indicator of Bone Metsastasis. Acta Radiologica 29(1988) Fasc. 4.445-449.

14. Wiesel S.W., Feffer H.L., Borenstein D.G. Evaluation and Outcome of Low-Back Pain of Unknown Etiology. Spine 13:6, 679 - 680. 1988.

15. Mellin G. Correlations of Spinal Mobility with Degree of Chronic Low Back Pain after Correction of Anthropometric Factors. Spine 12:5, 464-472. 1987.

16. Million R., Hall W. Haaiuk N. et al. Assessment of the Progress of the Back Pain Patient. Spine 7:204-212, 1982.

17. Evans J.H., Kagan A. The Development of a Functional Rating Scale to Measure the Treatment Outcome of Chronic Spinal Patients. Spine.11:3, 277-281. 1986.

18. Fairbank J.C.T. et al. The Oswestry Low Back Pain Disability Questionnaire. Physiotherapy. August 1980. Vol 66. No. 8. 271-273.

19. Uden A. et al. Pain Drawings in Chronic Back Pain. Spine 13:4, 1988. 389-392.

20. Leyshon A., Kirwan E., Wynn Parry C.B. Is it Nerve Root Pain? J. of Bone and Joint Surg. 62-B:119, 1980.

21. Saal J.A. Electrophysiologic Evaluation of Lumbar Pain. In: Failed Back Surgery Syndrome. Spine: State of the Art Reviews. 1986. White A.H. (ed) Hanley and Belfus Inc. p. 21- 46.

22. Hazard R.G. et al. Functional Restoration with Behavioural Support. A One-year Prospective Study of Patients with Chronic Low Back Pain. Spine 14;2, 1989. 157-161.

23. Mayer T.G., Gatchell R.J., Kishino N., et al Objective Assessment of Spine Function Following Industrial Injury. Spine.10:6, 482-493, 1985.

24. White A.A. Synopsis: Workshop on Idiopathic Low Back Pain. Spine. 7:141-149, 1982.

25. Dixon A. St. J. Diagnosis of Low Back Pain. Sorting the Complainers. The Lumbar Spine and Back Pain. Second Edition. Jayson MIV. (ed). Pitman Medical. 1980. pp. 135 -155.

26. Bernard T.N., Kirkaldy-Willis W.H. Recognising Specific Characteristics of Nonspecific Low Back Pain. Clin.Orthop. & Rel. Res. No. 217 April 1987. pp. 266-280.

27. Hansson T. et al. The Bone Mineral Content of the Lumbar Spine in Patients with Chronic Low Back Pain. Spine 10:2, 1985, 158-160.